AMENDMENTS TO THE CLAIMS

Please replace all prior versions, and listings, of claims in the application with the following list of claims, in which insertions are indicated by underlining and deletions are indicated by strikeouts or double bracketing.

1. (Currently Amended) A method for inhibiting lectin complement pathway (LCP) associated complement activation, comprising administering an effective amount of a mannose binding lectin (MBL) inhibitor to inhibit LCP associated complement activation in a subject with cellular injury, wherein the MBL inhibitor is a peptide, protein, or antibody or antigen-binding fragment thereof, and wherein the LCP associated complement activation mediates a cellular injury.

2. (Canceled)

3. (Original) The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with atherosclerosis.

4. (Original) The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with the pulmonary system.

5. (Original) The method of claim 4, wherein the MBL inhibitor is administered to the subject by an aerosol route of delivery.

6. (Original) The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with arthritis, myocardial infarction, ischemia, reperfusion, transplantation, cardiopulmonary bypass (CPB), stroke, acute respiratory distress syndrome (ARDS), systemic lupus erythematosus (SLE), lupus, or dialysis.

7. (Original) The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with ischemia.

8. (Original) The method of claim 2, further comprising administering to the subject a therapeutic treatment for treating an MBL mediated disorder associated with the cellular injury mediated by LCP associated complement activation.

9. (Original) The method of claim 8, wherein the therapeutic treatment is a drug.

10. (Original) The method of claim 8, wherein the therapeutic treatment comprises revascularizing a coronary artery.

11. (Original) The method of claim 10, wherein the revascularizing of a coronary artery is achieved by a method comprising percutaneous transluminal coronary angioplasty.

12. (Original) The method of claim 1, wherein a mammalian cell with a surface exposed MBL ligand is contacted with the MBL inhibitor.

13. (Original) The method of claim 1, wherein the MBL inhibitor binds to MBL.

14. (Canceled)

15. (Original) The method of claim 13, wherein the MBL inhibitor binds to a human MBL epitope.

16. (Original) The method of claim 15, wherein the human MBL epitope is a region of MBL which interacts with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

17. (Original) The method of claim 13, wherein the MBL inhibitor competes for binding to MBL with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

18-21. (Canceled)

22. (Previously Presented) The method of claim 13, wherein the MBL inhibitor is an antigen-binding fragment of an antibody.

23. (Previously Presented) The method of claim 22, wherein the antigen-binding fragment is selected from the group consisting of an F(ab')$_2$ fragment, an Fd fragment, an Fv fragment, and an Fab fragment.

24. (Previously Presented) The method of claim 22, wherein the antigen-binding fragment is of a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

25. (Original) The method of claim 13, wherein the MBL inhibitor is an antibody.

26. (Original) The method of claim 25, wherein the antibody is a monoclonal antibody.

27. (Original) The method of claim 26, wherein the monoclonal antibody is produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

28. (Original) The method of claim 25, wherein the antibody is a single-chain antibody.

29. (Original) The method of claim 25, wherein the antibody is a humanized antibody.

30. (Original) The method of claim 1, wherein the MBL inhibitor inhibits C3b deposition.

31. (Previously Presented) The method of claim 30, wherein the MBL inhibitor is an MBL binding peptide, protein, or antibody or antigen-binding fragment thereof, and inhibits C3b deposition with an EC50 of between 10^{-9} to 10^{-7} mol/L.

32. (Original) The method of claim 1, wherein the method further inhibits VCAM-1 expression.

33. (Original) The method of claim 1, wherein the MBL inhibitor binds to a mannose-binding lectin-associated serine protease (MASP) or mannan.

34. (Canceled)

35. (Previously Presented) The method of claim 33, wherein the antibody or antigen-binding fragment thereof is a monoclonal antibody or monoclonal antibody fragment.

36. (Previously Presented) The method of claim 33, wherein the antibody or antigen-binding fragment thereof is humanized.

37. (Previously Presented) The method of claim 33, wherein the antibody or antigen-binding fragment thereof is a single-chain antibody.

38. (Original) The method of claim 33, wherein the MASP is MASP-1 or MASP-2.

39. (Canceled)

40. (Previously Presented) A method for inhibiting cellular injury in a subject, comprising

administering an effective amount of an MBL inhibitor to inhibit cellular injury in the subject, wherein the MBL inhibitor is a peptide, protein, or antibody or antigen-binding fragment thereof.

41. (Original) The method of claim 40, wherein the cellular injury contributes to tissue injury associated with atherosclerosis.

42. (Original) The method of claim 40, wherein the cellular injury contributes to tissue injury associated with the pulmonary system.

43. (Original) The method of claim 42, wherein the MBL inhibitor is administered to the subject by an aerosol route of delivery.

44. (Original) The method of claim 40, wherein the cellular injury contributes to tissue injury associated with arthritis, myocardial infarction, ischemia, reperfusion, transplantation, CPB, stroke, ARDS, SLE, lupus, or dialysis.

45. (Original) The method of claim 40, wherein the cellular injury contributes to tissue injury associated with ischemia.

46. (Original) The method of claim 40, further comprising administering to the subject a therapeutic treatment for treating an MBL mediated disorder associated with the cellular injury.

47. (Original) The method of claim 46, wherein the therapeutic treatment is a drug.

48. (Original) The method of claim 46, wherein the therapeutic treatment comprises revascularizing a coronary artery.

49. (Original) The method of claim 48, wherein the revascularizing of a coronary artery is achieved by a method comprising percutaneous transluminal coronary angioplasty.

50. (Original) The method of claim 40, wherein the MBL inhibitor inhibits MBL deposition on a mammalian cell with a surface exposed MBL ligand.

51. (Original) The method of claim 40 or 50, wherein the MBL inhibitor binds MBL, MASP or mannan.

52. (Canceled)

53. (Previously Presented) The method of claim 51, wherein the antibody or antigen-binding fragment thereof is a monoclonal antibody or a monoclonal antibody fragment.

54. (Previously Presented) The method of claim 51, wherein the antibody or antigen-binding fragment thereof is humanized.

55. (Previously Presented) The method of claim 51, wherein the antibody or antigen-binding fragment thereof is a single-chain antibody.

56. (Original) The method of claim 51, wherein the MASP is MASP-1 or MASP-2.

57. (Original) The method of claim 51, wherein the MBL inhibitor binds to a human MBL epitope.

58. (Original) The method of claim 57, wherein the human MBL epitope is a region of MBL which interacts with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

59. (Previously Presented) The method of claim 51, wherein the MBL binding peptide, protein, antibody, or antibody fragment competes for binding to MBL with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

60. (Original) The method of claim 40, wherein the MBL inhibitor inhibits C3b deposition.

61. (Previously Presented) The method of claim 60, wherein the MBL inhibitor is an MBL binding peptide, protein, antibody or antigen-binding fragment thereof, and inhibits C3b deposition with an EC50 of between 10^{-9} to 10^{-7} mol/L.

62. (Original) The method of claim 40, wherein the method further inhibits VCAM-1 expression.

63-66. (Canceled)

67. (Previously Presented) The method of claim 40, wherein the MBL inhibitor is an antigen-binding fragment of an antibody.

68. (Previously Presented) The method of claim 67, wherein the antigen-binding fragment is selected from the group consisting of an F(ab')$_2$ fragment, an Fd fragment, an Fv fragment, and an Fab fragment.

69. (Previously Presented) The method of claim 68, wherein the antigen-binding fragment is of a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

70. (Original) The method of claim 40, wherein the MBL inhibitor is an antibody.

71. (Original) The method of claim 70, wherein the antibody is a monoclonal antibody.

72. (Original) The method of claim 71, wherein the monoclonal antibody is produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

73. (Original) The method of claim 70, wherein the antibody is a single-chain antibody.

74. (Original) The method of claim 70, wherein the antibody is a humanized antibody.

75. (Canceled)